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Volume XVII DECEMBER 1956 Number 12

DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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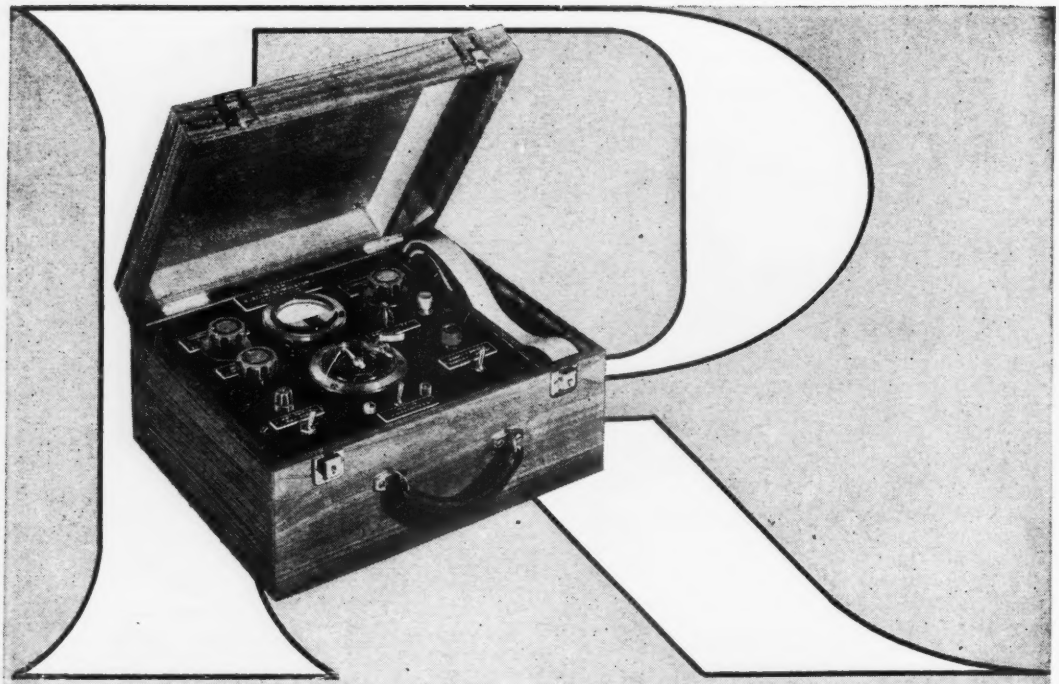

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DISEASES *of the* Nervous System

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

The Arnold-Chiari Malformation Without Spinal Dysraphia

IRA J. JACKSON, M.D., and GEORGE T. HOFFMANN, M.D.
Galveston, Texas

The Arnold-Chiari malformation is most frequently associated with a spina bifida. However, we have been able to find reports in the literature on 55 patients with this malformation but without a myelomeningocele or meningocele. In the absence of a spinal dysraphia, the reported causes of the Arnold-Chiari malformation consist of platybasia, scoliosis, craniovertebral anomalies, and in some instances, unknown factors. The clinical picture has varied, although intracranial hypertension is frequently present. We wish to record a 31-year old patient with quadriplegia due to an Arnold-Chiari malformation unassociated with increased intracranial pressure or a myelomeningocele.

The deformity known as the Arnold-Chiari malformation was so named by Schwalbe and Gredig in 1907,[1] resulting from their description of four patients with associated myelomeningoceles. The essential features were outlined by Arnold[2] in 1894, and by Chiari[3] in 1895. There is a caudal tonguelike prolongation of the cerebellum which overlays and is firmly bound down to a greatly elongated medulla oblongata in the upper part of the cervical spinal canal. When these two neural components are dissected apart, there is an elongated fourth ventricle with a tuft of choroid plexus frequently found occupying its most caudal portion. The lower cranial and upper cervical nerves are generally elongated and make their exit from the dural canal in a cephalad fashion.

There are two theories as to the pathogenesis of the Arnold-Chiari malformation. Many authors[1,4,5,6] favor the traction theory, in which there is tethering of the spinal cord at the site of a vertebral defect. The spinal cord is stretched as growth of the body continues and therefore the normal ascent of the cord is inhibited. On the other hand, Dorothy Russell[7] believes it probably occurs at an early stage of embryonic development independent of any myelomeningocele or vertebral defect.

This malformation was first reported unassociated with a spina bifida in 1938.[8] In reviewing[4,8-26] the 55 patients with Arnold-Chiari malformation unassociated with a spinal dysraphia one finds the proposed etiology to be variable. Twenty-four patients had platybasia, 10 Klippel-Feil syndrome, and others scoliosis, hemivertebrae, spina bifida occulta, and syringo-myelia. There was no pathogenesis reported in many of the cases. The ages of the patients ranged from the newborn to 60. The sexes were fairly equally distributed. Only 37 of the 55 patients were so recorded that their symptomatology could be adequately interpreted. The symptomatology was referable to increased intracranial pressure, the cerebellum, the lower cranial nerves, the spinal cord or any combination thereof. Thirty patients had cerebellar symptoms; in 25 it was related to increased intracranial pressure; in 23 to the lower cranial nerves and in 22 to the spinal cord. Ten patients exhibited the most frequent combination of symptoms in which all four groups were involved. Six patients exhibited symptoms referable to increased intracranial pressure and the lower cranial nerves, and another six patients to a combination of lower cranial nerves, cerebellar and spinal cord. None had just cerebellar symptoms or only lower cranial nerve involvement. Only three patients presented

Division of Neurological Surgery, Department of Surgery University of Texas Medical School—John Sealy Hospital.

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symptoms solely of increased intracranial pressure while 14 patients had no such signs or symptoms. Two patients had cerebellar signs, five had just nystagmus. The 17 patients presented by Gardner and Goodall[22] could not be included as the author gave no breakdown of symptoms but they did state that the only purpose of the operation was to relieve increased intracranial pressure.

The duration of symptoms also varied. In 26, there was a gradual onset ranging from three months to 16 years, with the great majority occurring prior to three years. Only seven patients were reported with symptoms of longer than three years duration. One patient had a 16-year history.

The diagnosis was made by myelography in eight patients and by pneumoencephalography in nine.

Fifty-four of the 55 patients were operated upon. The postoperative results were not recorded in six patients. Thirty-seven patients (77%) were considered improved, while five (10.5%) were not. The postoperative mortality was 12.5% (six patients). Three patients required more than one operation. The procedure essentially consisted of a decompression.

Case Report

N.H.N. (N.H.: No. 2666-I), a 31-year old white male was admitted on the neurological service at John Sealy Hospital on July 28, 1954, with the following history:

The patient was apparently well up until August of 1939, when he fell off a colt and landed on his right shoulder. He was apparently unconscious for about an hour and then made an uneventful recovery. However, within the following few months his mother noticed that his right shoulder drooped and that his shoulder blade protruded. A scoliosis was noted in November of 1939. About five months later he became aware of progressive numbness of his right hand.

On November 8, 1940, he was admitted to a University hospital elsewhere for two weeks with a scoliosis and a right hemiplegia, more pronounced and associated with atrophy in the upper extremity. There was anesthesia of his right fourth and fifth finger. A diagnosis of scoliosis and lordokyphosis was made and a long body cast applied.

The patient was readmitted to that hospital for two weeks on January 31, 1941. The body cast was removed. They noted a sensory loss on the right side from the fifth cervical down, and a right hemiplegia.

He was again readmitted on June 15, 1941, for five days. He was still able to get around at this time. The patient was unable to sweat on the right side of his face and body. Shortly after leaving the hospital he began to have episodes of a cramping-like pain in his back. There was weakness of both legs demonstrated by his progressive difficulty in walking. On August 11th and 16th, 1944, he had a severe cramp in his back and was unable to walk.

He was again readmitted to the same hospital on August 26, 1944, for almost six weeks. At this time the attending physicians found bilateral long tract sensory signs, bilateral pyramidal tract signs, and atrophy of both upper extremities including the hands. These deficits were all greater on the right than the left. They reported that nystagmus and optic atrophy were now present. X-rays of the skull, foramen magnum, and cervical spine were considered normal.

His fifth admission to that hospital for ten days was on February 6, 1945. He now had no movement of his right hand.

The patient stated that he had episodes of loss of vision lasting for 10 to 15 minutes in 1942, 1945, and in 1949.

By 1947, he had little movement of his right side but was still able to walk with the aid of a cane. He became aware of more weakness of his left leg in the following year. There were several momentary attacks of severe hyperextension of his trunk with tingling in his face.

The patient had been working as a welder as late as 1943, and for the next seven years was only able to care for his chickens. He had become a wheel chair patient by 1951. In 1952, the weakness of his left hand became so pronounced that he could only do very few and simple things for himself.

The family history and his own past history are non-contributory. His parents and five siblings are alive and well. One brother died at the age of 15 by accidental electrocution.

Our examination in August of 1954, revealed a very personable young man confined to a wheel chair because of his quadriplegia. He was able to void and have bowel movements with no difficulty. Both upper and lower motor findings were more pronounced on the right than the left. The hands were markedly atrophic and had extreme flexion contractures. All four extremities exhibited atrophy and fibrillations but this was more pronounced in the uppers. Shoulder movement was the only function he could perform with the right upper extremity. There was a slight amount of flexion and extension of the elbow on the left, but no hand motion. He was able to flex, extend, and abduct his hips, again greater on the left than on the right. There was very slight movement of the left ankle and knee, and none of the right. One examiner observed a right Horner's syndrome, but later this was not evident. There was a pronounced cervico-thoracic kyphoscoliosis. He had spasticity of his lower extremities with bilateral Babinskis. The deep tendon reflexes were absent in the upper extremities and in both knees, while the ankles presented clonus. Sensibilities to pain, touch, vibration and discrimi-

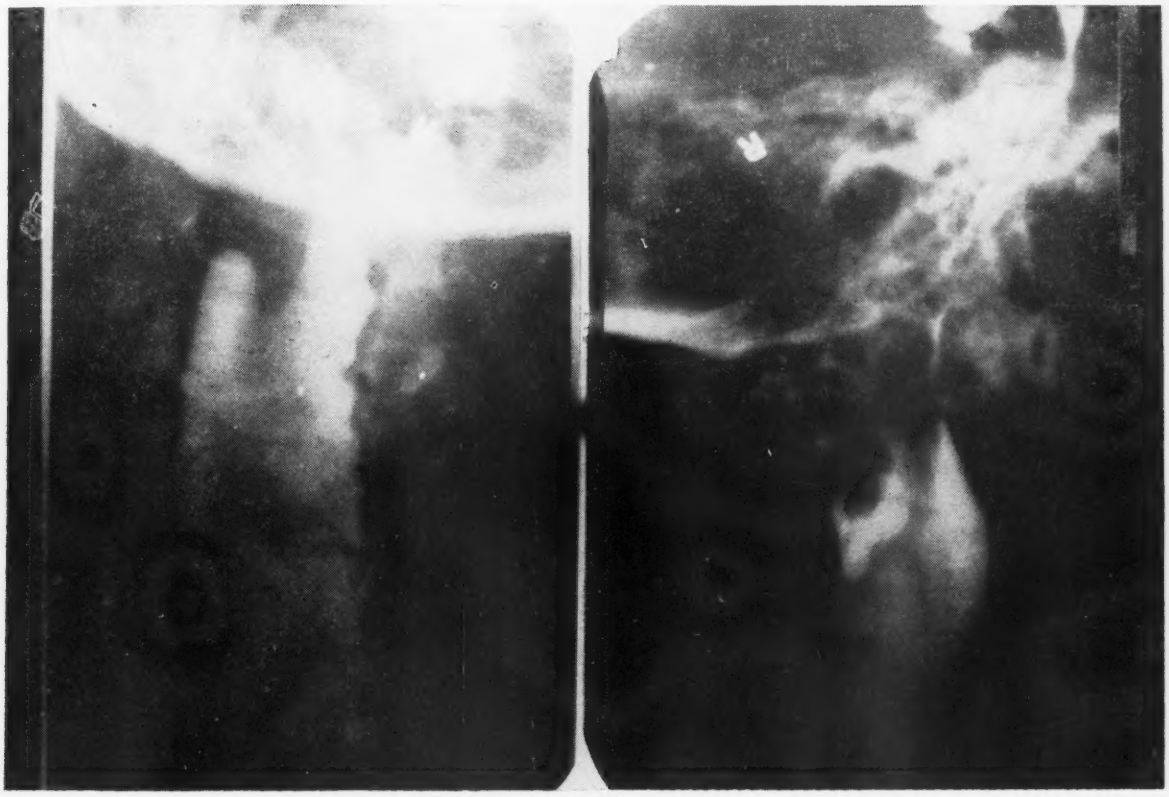

Figure 1. Myelogram of patient in head down position showing pedunculated defect caused by Arnold-Chiari malformation in A.P. and oblique view.

nation were absent on the right from the angle of the jaw down, and on the left from the clavicle with bilateral sacral sparing. Nystagmus was greater on left lateral gaze. The only evidence of any fifth nerve involvement was a questionable diminution of sensation of the first division on the right. The tongue showed some fibrillations, but no atrophy. The spinal fluid protein was 158 mm's per cent. The electroencephalogram was normal. Roentgenograms of the skull, cervical, thoracic and lumbar spines were taken many times only revealing essentially a kyphoscoliosis. There was no x-ray evidence of a foramen magnum lesion, platybasia, or a spina bifida occulta.

A pantopaque myelogram revealed a block at C-2 with a pedunculated type of lesion (Fig. 1).

On August 23, 1954, a cervico-suboccipital decompression was performed under general anesthesia in the sitting-up position. After the dura was opened, there was a pedunculated mass arising from the posterior aspect of the spinal cord at about the level of the first and second cervical. The tip of this mass was cystic. The mass was biopsied and shown to be cerebellum. The spinal cord in the upper cervical area appeared compressed and quite thin. The nerve roots made their exit in a cephalad manner. The cerebrospinal fluid flowed freely from above on each side of the mass but none could be seen coming out of the foramen magendie, wherever it might have been. An adequate decompression was effected by leaving the dura open and placing a piece of gel film over the large vent. The remaining wound was closed in layers without drainage.

The patient remained in the hospital until January 9, 1955, receiving intensive physical therapy. He received a fractured humerus while being given physical therapy. There was extensive osteoporosis resulting from prolonged disuse. However, he continued to show improvement in his neurological deficit up until the time of his discharge from the hospital.

When seen nine months later, he had bilateral hip flexion and abduction. There was some flexion and extension at the knees, but no motion of the ankles or feet. He was able to flex and abduct his right shoulder. There was good movement of the left upper extremity including the shoulder, elbow, supination, pronation and even flexion of the thumb. There was some tightening of the abdominal muscles for the first time on raising his head. He was able to sit upright in a chair without support for the first time in years. The sensory level was now at the seventh cervical. The nystagmus is less pronounced. He was able to aid himself in eating.

It would appear that the Arnold-Chiari malformation probably occurs during the early embryonic development independent of any traction of the spinal cord. If the traction theory were wholly responsible for such a malformation, it would occur more fre-

quently and would not be found in the absence of a tethered spinal cord. Spinal dysraphia and the elongation of the hindbrain and cerebellum are merely independent defects in development. The reported causes of this malformation unassociated with myelomeningocele as platybasia, craniovertebral anomalies are merely other embryonic developmental defects and not etiological factors. In those patients unassociated with spinal dysraphia, the clinical features fall into four general categories: increased intracranial pressure, lower cranial nerves, cerebellar and spinal cord. Frequently, there is a combination of these four groups of symptoms. Our patient's symptomatology is unique in that there is but one other recorded case with only cerebellar and spinal cord signs. The diagnostic procedures most frequently employed are myelography and pneumoencephalography. A pedunculated filling defect is visualized just below the foramen magnum. Surgical therapy, being the only form of treatment, has proven worthwhile (77% were improved) and should be instituted as soon as possible. As in our patient, this consists of an adequate suboccipital cervical decompression. The diagnosis should be made long before quadriplegia occurs.

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Monopolar Electro-Convulsive Coma Therapy for "Hard Core" Cases

JOSEPH EPSTEIN, M.D., and BRIAN C. CAMPDEN-MAIN, M.D.

Introduction

There is considerable evidence in the literature that prolonged electrocerebral stimulation which produces coma or sleep may be an effective therapeutic agent in "hard core" cases. By "hard core" cases we mean those which have failed to respond to conventional ECT and have already received insulin coma without success or because of their severity were slated to receive insulin coma. The techniques by means of which electrocerebral stimulation produces sleep or coma vary. Until recently the only currents used have been either AC or unidirectional and the active electrodes are bipolar and placed bilaterally, in various positions at the frontal, parietal or temporal areas of the head. Bursts of current are introduced depending on the tolerance of the patient and the electrodes are cautiously moved from the frontal regions downward toward the temporal regions, or vice versa, depending upon the cardiorespiratory tolerance of the patient. The resultant sleep states have been designated as electronarcosis[1,2] or electric coma.[3] The technique is not simple; it requires an experienced team to administer the current, manipulate the electrodes and watch the vital signs of the patient. In many instances the treatment seems quite rough; respiration tends to be stridulous and the patient too hyperkinetic. These methods, although showing clinical promise have not been widely accepted because of the difficulty of the technique.

Our present research was stimulated by the belief that if the method of induction of electrocoma were improved and simplified, it could be introduced to larger segments of psychiatric practice and made possible for ambulatory treatment. We believe we have accomplished this by utilizing the monopolar methods of Epstein.[4,5] Applying these techniques with slight modification, we found that convulsive and subconvulsive stimulation could be effected with minimal currents and that respiratory difficulties and excitement can be eliminated. The treatment is not complicated and the same physician who manipulates the current also handles the monopolar electrode.

From Pinewood, Katonah, N. Y., and Central State Hospital, Petersburg, Va.

Technique

The descriptions to follow refer exclusively to the use of the Reiter RC47C machine. Patients before being accepted for this treatment should have the same preliminary work-up; including E.K.G., etc. as for any form of ECT. We do not use muscle relaxants. We have not used intravenous barbiturates except to allay anxiety. In such cases, we inject 3¾ to 7½ grs. Sodium Pentothal in a 10% solution immediately prior to the treatment. About one-half hour before treatment the patient receives 1/25 grain of atropin sulphate either under his tongue or by hypo. This is given not only to control salivation, but to avoid cardiac arrest which may occur temporarily following convulsive electrostimulation. Treatment is administered to the patient either in his own bed or upon a treatment table. The physician administering the current sits on a stool behind the head of the patient. In his right hand he holds the insulated handle of the active (negative) monopolar electrode. The machine is placed on a table convenient to the left hand of the operator. Since the entire treatment is performed by manual manipulation of one electrode, the physician can with ease operate the controls on the machine and control the electrode without assistance. A cuff electrode (positive) is now applied to the right forearm of the patient, and a small piece of rubber sheeting (for purposes of insulation) is placed between the wet cuff electrode and the shirt of the patient. The patient's arms are then folded across his chest and we are ready to start the treatment. This is initiated by inducing a grand mal convulsion by the monopolar technique of Epstein.[5] It consists of applying the No. 5 current of the

Reiter machine at 10-17 ma. intensity for 3 to 10 seconds. With the current pre-set, the active electrode is applied to the right frontal region near the vertex and about one inch to the right of the midline. A typical convulsive seizure ensues within 3 seconds or longer, at which point the current intensity is reduced by about one-half or just strong enough to prevent excessive clonic movements.

At about 35 seconds, the current intensity is reduced to about one ma., while we await the return of respiration. From this point on we commence the technique of induction of the monopolar electric coma. An assistant sitting at the side of the patient holds in place an ordinary stethoscope and notes the rate, rhythm and quality of the heart sounds. As treatment progresses, the rate tends to be rapid—generally from 140-170 per minute; its quality is good. Any sudden drop in the rate or irregularity in rhythm may be an indication to reduce the current or discontinue the treatment. We have never found it necessary to insert an air-way or use oxygen. An emergency set-up making these available is, of course, routinely on hand as for any form of ECT. At the beginning of treatment we insert a dental roll between the teeth. This keeps the teeth separated, absorbs excessive saliva, and leaves an air space. During the minute or so after the termination of the convulsion and while the patient is breathing and comatose, we reset our controls to induce electrosleep. The modulation control knob is turned to the left to about 12 o'clock; the ratio-control knob is turned all the way to the right and the electrode is applied about one inch to the right of the sagittal midline and an inch in front of the vertex. With the ratio knob to the right, the current intensity is brought up to the maximum possible consistent with free breathing (generally ½ to 2 ma.); then the red button is depressed (this increases the current by about twice or more over the basic intensity to about 4 to 5 ma.). Our aim in delivering this "burst" of current is to produce about 6-8 clonic movements of the lower extremities over a period of 3-5 seconds. These "bursts" of current are delivered 5 to 6 times a minute at the end of an audible respiratory phase—inspiration or expiration. It is absolutely necessary at the inception of the induction of electric coma to produce high enough "bursts" with the red button to have 6-8 clonic leg movements each time. When the button is allowed to recoil, we expect to find the ratio knob so adjusted that the basic current will permit the patient to breathe without stridor. If this is not immediately possible, we remove the electrode to allow completion of the interrupted breathing cycle at zero current. Immediately at the completion of inspiration, the electrode is replaced and simultaneously the red button is pressed to bring about a "burst" of stimulation. If on releasing the red button, respiration should continue difficult, the electrode is lifted thereby dropping the current to zero for a second or two to allow return of respiration. The removal of the electrode at every expiration is repeated as long as necessary to insure proper breathing. By doing this we are able to secure high enough current values to induce coma without worrying whether the base current is allowing respiration to proceed. As this goes on we soon find that the patient's tolerance to the current increases, and with experience, the ratio knob and current intensity control can be adjusted so that as treatment proceeds, the patient is able to breathe with the current on, and he remains asleep with lower "bursts" of current, or without the necessity of any red button bursts.

With the above technique and for a period of four minutes the electrode is held alternately in front of the vertex and to the right of the midline and its corresponding area to the left of the midline. The position of the electrode is changed every half minute from one side to the other. As it is moved, it should be dipped in electrolyte solution before being replaced. During this process, the current is broken and the patient takes a deep breath. This is desirable and is an important part of our technique.

After four minutes, the monopolar electrode is shifted from the region of the vertex to the area in front of the right ear and for one minute or longer, it is slowly moved back and forth along the hair line from in front of one ear to the other. About 3 to 5 ma. of current is generally well tolerated while patient

continues to sleep. By thus sweeping the current from side to side, we are actually spraying every part of the cerebrum with current very much in the shape of a "V," with the apex at the foramen magnum or medulla and the current passing in a wide triangular area with the base between the ears. After five or six minutes, the current is turned off and the treatment is ended.

With the monopolar technique there is no hyperkinesis. Should this appear it is an indication of undertreatment and the current values or "bursts" are at once increased to eliminate any such tendency. Occasionally 10 to 15 seconds may elapse before this hyperkinesis is overcome by proper increase in current. The patient will deny having felt the treatment but at times he will show a certain diffuse anxiety. Recently a patient told us on awakening that she had a nightmare in which she had to run very fast to catch up with a crowd of people who seemed to be running faster than she. She stated that she was running so fast that she was out of breath, and as she ran, something was banging on her head. (The bursts of current cause interference with respiration and may give the feeling of banging on the head and being unable to breathe.) This dream does seem to indicate the ability of the patient to record stimuli on a subconscious level and to translate them as a kind of warning or threat to survival. Perhaps we may find in this a clue to the intense anxiety and fear which certain patients develop toward continued ECT in any form. Although patients are mentally clear on awakening and show no confusion, there is nevertheless some variation in reactions. Some patients awake quietly and then go back to sleep. Others remain asleep for from 5 minutes to half hour. Occasionally a patient awakes at once and begins talking. Some patients awake with a pleasant attitude.

We prefer not to use intravenous barbiturates. We were interested in observing the effects of the electrocerebral stimulation per se without any possible alterations due to depressant drugs—and further— we felt that in some instances the patient remains asleep as a result of the anesthetic and we do not really know how much sleep has been induced by the electric current. There may be reason to believe that preliminary intravenous anesthesia may contribute sufficiently to the induction of electric coma, as to warrant its more general use, and it is possible that in the future we may use Pentothal as an adjunct to the induction of electrocoma.

This report concerns itself with the clinical findings in a series of 40 patients. Twenty cases were treated at Pinewood and twenty at the Central State Hospital, Petersburg, Va. The technique described above was developed after several months of independent research with various currents and various areas of stimulation. As already mentioned, all cases selected were considered "hard core." Their duration varied from 7 mos. to 16 years. All had received previous ECT or insulin coma, and were either unimproved or had relapsed after improvement. Some had had 2 to 4 courses of ECT with no improvement. Most of the patients were schizophrenics or paranoid involutional psychotics. Several were psychotics showing atypical affective reactions. A significant number of patients who had failed to improve with other methods were recovered or much improved with electrocoma. Another interesting finding was that we were unable to predict which patient would or would not benefit from electrocoma. Duration of illness did not afford a clue to the possible outcome. In the Central State Hospital, five of the patients who were much improved had a duration of illness of over 5 years and one had been ill for 16 years.

Another interesting fact was that several patients showed sudden improvement or recovery after only one or two electrocoma treatments. In such instances the patient had had a series of about 20 conventional ECT's with no improvement whatever. Upon changing the technique to electrocoma, it was found that one or two additional treatments resulted in spectacular recovery. As an example of this we may cite one case of a 60 year old male who was suffering from a paranoid type of involutional psychosis. He had had 28 conventional ECT's with both AC and unidirectional current methods without improvement. He recovered suddenly after two electrocomas.

A similar result was obtained in a 20

year old paranoid schizophrenic who had previously received 17 ECT's consisting of alternate AC and unidirectional methods of convulsive seizures with no improvement. The treatments were changed to electrocoma and after six treatments he made a complete recovery. There was one death. Autopsy showed gross fatty infiltration of the visceral pericardium and of the myocardium; the right coronary artery was markedly sclerosed. The myocardial degeneration was so severe that the patient might have died of any strain.

In the chart below we consider those patients "much improved" who were well enough to return home and make a social recovery. By "improved" we mean those patients whose mental condition and behavior were improved but who remained hospitalized.

TABLE OF RESULTS

	No. Pts.	Recov.	Much Impr.	Impr.	Unimpr. or Relapsed
Pinewood	20	5	10	1	4
C.S.H.	20		8	2	10
Total	40	5	18	3	14
Percentage		12.5%	45%	7.5%	35%

Discussion

Until we know more exactly how ECT works, it will be necessary to explore such modifications of techniques as can raise the number of recoveries or improvements above that obtained with conventional methods. The induction of electrocoma as described here consists of two phases of stimulation. The first introduces a grand mal seizure and lasts about 45 seconds; the second phase consists of subconvulsive stimulation in alternate low and high phases, resulting in coma, and lasting about 6 minutes. During the latter period, the greatest stimulation is applied sagittally to the cerebrum, passing from the medulla where the current from the indifferent electrode enters to the point of application of the active electrode. This specifically stimulates the various thalamic nuclei, the reticular activating substance, and the pons and medulla. Toward the end of coma, by sweeping the monopolar electrode across the front of the head, we actually produce stimulation of the entire cerebrum. It is interesting to note that with this method there appears to be no significant residual confusion following the treatment.

This confirms the "shock-countershock" theory of Leo Alexander[6] and lends clinical evidence to the belief that there are stimulative effects on cerebral tissue when subconvulsive currents are applied. This also confirms Goldman who found that EEG changes were less pronounced in electrocoma than with conventional ECT.[7]

As further modications in cerebral electrotherapy are evolved, we can arrive at some general conclusions. First, we must admit that as yet we are ignorant of the exact changes which the electric current produces in its passage through the brain. Evidence is accumulating that the conventional convulsive current whether AC or unidirectional produces its greatest benefit in psychotic depressions; i.e., the manic depressive states and the involutional psychoses. Additional evidence is accumulating to show that in schizophrenia,[8,9,10] electric sleep seems to bring about changes akin to those produced by insulin coma. The production of electric sleep further has an analogy to insulin sleep in that it apparently first stimulates and then depresses the cerebrum beginning with the cortex and invading the deeper and older structures later, and with prolonged treatment posing a threat to the whole autonomic system, as does insulin. We are convinced that the answers to these problems will be solved in the domains of electro-physiology and neuropharmacology.

The main purpose of this paper has been to introduce a new technique, which can be easily acquired, for the induction of electric coma. Confidence in the method and mastery of the technique can be facilitated by observing its administration in clinics where it is in use. Our clinical report is only preliminary and many more cases will have to be treated and studied against controls in order to arrive at statistically significant conclusions. There are, however, many favorable reports in the literature concerning this form of treatment for schizophrenia and other psychoses, and our results lend further support to the merit of the method.

Summary

We have described a new technique for inducing electrocoma treatment. It is performed by application of a monopolar electrode to certain regions of the head. The indifferent electrode is a cuff applied to the right forearm.

The monopolar technique for inducing electrocoma is less difficult than any of the bipolar methods now in use. Its clinical results seem to parallel or excel those of electro-coma produced with bipolar techniques.

This treatment is far easier to administer than insulin coma. It is less hazardous and takes less time. It is suitable for ambulatory treatment since patients awaken shortly after the treatment and are in good contact, showing no exhaustion or confusion.

Monopolar electrocoma therefore recommends itself for "hard core" cases, and for consideration as a method to be used before resorting to insulin coma.

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N-Methyl-a, a-Methylphenylsuccinimide in Psychomotor Epilepsy

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Preliminary observations regarding the anticonvulsant action of N-methyl-a, a-methylphenylsuccinimide or PM 396 (Parke, Davis and Company) suggested a beneficial effect in petit mal and psychomotor seizures. Further study in patients with petit mal and a smaller group of patients with psychomotor epilepsy confirmed this impression.[1,2] The present article deals predominantly with an evaluation of the effect of PM 396 medication in psychomotor epilepsy, although the results in some other types of epilepsy have been included.

The approach utilized was to take a group of patients, who had been well studied clinically and with electroencephalography and who had been treated for a considerable time with different drugs in various combinations without ever having been adequately controlled. PM 396 then was added to the last medication the patient was taking. This represents rather unfavorable conditions to start with, of course. On the other hand, many effective medicines for the control of epilepsy are at present available, whose reliability and side effects are well known. It could be argued that a new medication would especially prove its value, when successful, where other drugs have failed because of lack of control or excessive side reactions. Also, it is not easy to obtain sufficient material in psychomotor epilepsy to study the effect of a new medicine in an as yet untreated group of patients.

The total number of patients receiving PM 396 was fifty, ranging in age from four

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to fifty years. There were twenty-one male and twenty-nine female patients. Fourteen, however, had to be excluded from our summary for various reasons. Seven of these (four with psychomotor epilepsy, one with grand mal, one with petit mal and one with Jacksonian seizures) took the drug for less than seven days. They claimed no benefit and were disturbed by the side effects (drowsiness in five instances, a rash in one case and ataxia in one). Four patients (three with psychomotor epilepsy and one with grand mal) were in general discouraged with their illness and the many fruitless attempts at control. They did not keep records, took their medicine irregularly, returned at odd intervals and were unreliable in their reports. In one patient with psychomotor epilepsy it was impossible to evaluate the result, because of his associated psychiatric condition, but he seemed to derive some benefit. Another patient with psychomotor epilepsy, who initially responded well, became pregnant and developed psychomotor status in the third month of pregnancy, while taking PM 396. She did well again, subsequently, on the same medication. The last patient of this group of fourteen, suffering from focal frontal lobe seizures, reported an increase in the number of seizures and discontinued the medicine after a few days.

In considering the results of this study, the fact that this group of fourteen patients has been omitted has to be taken into account. It is, however, extremely difficult to evaluate these patients. Partly, this is undoubtedly due to the fact that in a series of patients with psychomotor epilepsy disturbing personality factors often play a considerable role. In actual practice, however, the results in this group probably have to be considered as a therapeutic failure.

The following report, then, deals with a group of thirty-six patients. For the classification of seizure pattern we have tried to adhere as closely as possible to Gibbs' combined clinical and electroencephalographic approach.[3] Each patient had from one to four electroencephalograms with a total of 62 recordings in 36 patients, in the majority both a waking and a sleeping record. The electroencephalographic changes with PM 396 and other medication will be the subject of a separate report. Twenty-three patients had psychomotor epilepsy; thirteen of these occasionally also had generalized seizures, always preceded by their particular aura, and in twenty a temporal lobe focus was present in the electroencephalograms, while in the remaining three the electroencephalograms were normal, but the clinical seizure pattern was, undoubtedly, of a psychomotor nature. Additionally, there were five patients with petit mal (three had had occasional grand mal attacks, but these had been controlled with other medication). These five patients all showed a paroxysmal generalized three per second spike and wave pattern on the electroencephalogram. Two patients had grand mal seizures (with a normal electroencephalogram), two had myoclonic seizures and grand mal attacks (with a generalized multiple spike and slow wave discharge), three patients had focal minor seizures (two with a unilateral spike focus in the parietal lobe, and one with a unilateral spike focus in the frontal lobe), and one had myoclonic seizures (with a focal multiple spike and slow wave discharge in one frontal lobe).

TABLE I—*Number of Patients and Number of Drugs They Received at Different Times Before PM 396 Treatment.*

Number of Drugs	2	3	4	5	6	7	8
Number of Patients	4	8	6	11	5	1	1

This group of thirty-six patients had previously been treated with two or more of the following medications in various combinations: phenobarbital, Mebaral,[R] Hibicon,[R] bromides, Phenurone,[R] Carbromal,[R] Dilantin,[R] Mesantoin,[R] Mysoline,[R] Tridione,[R] Paradione,[R] Milontin[R] and Diamox.[R] (Table I.)

The results of treatment with PM 396 are recorded in Table II.

TABLE II—*Results With PM 396 (See Text)*

	Total	Improved	Unimproved
Psychomotor seizures	23	15	8
Petit mal	5	4	1
Grand mal	2	*	2
Myoclonic seizures	3	2	1
Focal frontal lobe seizures	1	0	1
Focal parietal lobe seizures	2	2	0

The initial criterion for improvement was a reduction in seizure frequency of more than 50%. This is an arbitrary dividing

line. In practice, however, there was little difficulty in deciding how the individual patient had to be considered. The patients who did improve showed a marked reduction in seizure frequency as evidenced by the fact that this group of twenty-three fell from a total weekly seizure frequency of 144 (taken as an average over the preceding three months, while on other medication) to a total weekly seizure frequency of 9 (with PM 396 added to this medication). The thirteen patients who did not improve did not get any benefit, or so little as to be dubious and certainly below the 50% dividing line.

The follow-up time, during which the patients were carried on PM 396 medication, is recorded in Table III.

TABLE III—*Duration of Treatment (in Months) With PM 396.*

	Number of Patients	Minimum	Maximum	Average for Group
Improved patients	23	1½	11½	5½
Unimproved patients	13	½	11	4½

Side Effects—An important feature was the frequency with which side effects appeared with PM 396 medication. These did not occur in fourteen of the thirty-six patients, but twenty-two reported various disturbing sensations and reactions. Some had multiple complaints, others only a single side effect. The different complaints are tabulated in Table IV.

TABLE IV—*Nature of Side Effects in 22 Patients—of a Total of 36.*

Drowsiness	11	Loss of appetite and weight	3
Ataxia, dizziness	8	Constipation	3
Irritability and nervousness	5	Headache	3
Blurred or double vision	4	Diarrhea	1
Nausea	4	Rash	1

No relation could be established between side effects and age, dosage or type of seizure. All patients were started on 0.3 gm per day and the dosage was slowly increased, depending on the nature of the side reactions and the effect on seizure frequency. Final dosage ranged from 0.3 to 1.5 gm. a day with an average of 0.9 gm. The severity of the side effects varied considerably. One patient took only one capsule which, according to her, "put her in the hospital for three days." It is significant that only one patient of the improved group stopped his medication, because of the severity of side reactions. All others were willing and able to continue on their medication, even though, occasionally, dosage had to be reduced.

Summary — N-methyl-a, a-methylphenylsuccinimide was administered to a group of fifty patients with epileptic seizures. These patients had been well studied with electroencephalography and had been treated for a considerable time with various other medications without ever achieving adequate control of their seizures. Thirty-two suffered from psychomotor seizures. In nine of these thirty-two the effect of the new medicine could not be properly evaluated, because the patients discontinued the drug after a short time or were unreliable in their reports. In fifteen of the thirty-two seizure frequency was reduced significantly, more than 50%. In eight no improvement was noted. N-methyl-a, a-methylphenylsuccinimide thus seems to have a definite value in the treatment of psychomotor seizures.

In some instances, patients with petit mal, focal minor seizures or myoclonic seizures also reported a definite beneficial effect.

Undesirable side effects were frequent, though in the improved patients rarely of such severity that dosage had to be reduced or the medicine discontinued. Drowsiness, dizziness and ataxia occurred most frequently.

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Medicolegal Aspects of the Doctor-Patient Relationship

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Since the existence of every individual continues to have legal significance prior to his birth and even after his death, each of us is subject to unceasing legal obligations and is correlatively invested with certain rights and duties.

As a particular segment of society disciplined in the nature of their calling, definite legal essentials fall to the responsibility of physicians in their tasks of diagnosis and treatment. The meagre, and in many instances, the still almost negligible amount of medical student training in the legal aspects of doctor-patient relationships urges this line of inquiry which touches upon the doctor's prerogatives as well as his responsibilities.

Only after an ordered understanding of the pertinent legal implications which bear upon him, may the physician be enabled to act with confidence and be prompted in his medical decisions and judgment without coming into conflict with the rules of law concerned therewith.

The medical man's job requires that he give treatment to his patient in a skillful and non-negligent manner. To that end the doctor is duty bound to keep abreast of the times, and departure from recognized methods in diagnosis and treatment, if it results in injury, will render the physician liable to the patient in damages, irrespective of how well intentioned the doctor has been; and this responsibility exists whether the patient paid for the treatment or was treated free of charge.

Because of his position of trust, it is the physician's duty to act in good faith and to advise the patient as to the possibility of benefit or cure, or of the necessity of other type of treatment. (Benson v. Dean, 232 N. Y. 52). If the physician frankly notifies his patient of his want of professional skill, or if the patient becomes otherwise aware of this fact, the patient will not succeed at law if he complains of this lack of which he knew. (Lorenz v. Jackson, 34 N. Y. S. 652.)

A physician is not liable for a bad result or for failure to cure the patient as long as he has used the requisite degree of skill. However, he may agree to achieve a certain result or a cure, and if he fails, he becomes legally liable for a breach of contract, even though he knew at the time he entered into the agreement, that he could not cure, but the patient thought the physician could. (Colvin v. Smith, 92 N. Y. S. 2nd 794.) If the physician enters into such an arrangement unwisely he becomes liable if he fails to produce the specific result, on the ground that he failed to fulfill the agreement he had made. Although a patient has a right to be protected from negligent medical care, his complaint that a preparation which has cured others, failed to cure him or has caused him harm, will not render the physician liable in damages. (Parker v. State, 105 N. Y. S. 2nd 735.)

Whenever the patient or members of his family suggest dissatisfaction, or without adequate reason, complain to the attending physician, or where there appear to be a lag in the patient's improvement, or where some unanticipated situation or reaction is taking place during the treatment, it will protectively serve both patient and doctor to bring in an independent consultant. Moreover, it becomes a moral duty in difficult cases for an attending physician to refer his patient for consultation to the specialist because of latter's greater qualification in the skills of the appropriate medical situation. This exercise of prudence by the attending physician in obtaining the benefit of consultative collaboration, particularly where the patient's medical progress is not promising, will tend to create a favorable influence upon a jury in a legal action against the physician, to show that the latter's reaching out for impartial consultation was an exercise of a proper degree of care, while the patient was under his treatment. As an incidence

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From a lecture delivered by invitation of the Education Committee of Fordham Hospital, New York, to the House and Resident Staffs.

to this situation, the patient is responsible for the consultant's fee when the latter was called in by the patient's attending physician, either with or without consent or knowledge of the patient. This proposition of law was made clear in the case of Tobey vs. Nelson, 270 N. Y. S. 201.

A physician is legally as well as ethically bound to remain in attendance upon a patient who is in a critical condition, and can only discontinue his services to the patient upon the latter's consent, or after having given the patient reasonable notice to get another physician. (Becker v. Jaminski, 15 N. Y. S. 675.)

An interesting question arises in situations where a physician is subsequently called upon to treat a patient for a condition initially treated by another. A case in point is where a chiropodist first negligently treated a patient's foot causing it to become infected, and thereafter the physician who succeeded the chiropodist negligently treated the limb causing further aggravation of the condition. The court held that the chiropodist, as the initial tort-feasor (wrong-doer) becomes liable for the patient's totally sustained damage. (Prines v. Ross, 123 N. Y. S. 2nd 702.)

A surgeon is entitled to rely on the hospital nurse and has a right to assume that its nurses, not hired by him, are competent. In the absence of evidence that the nurse acted under his immediate supervision and control, unless in the exercise of ordinary prudence injurious effects of her negligent acts might have been prevented, a surgeon performing an operation in the hospital is not chargeable with negligence of its nurse personnel. The same rule applies respecting the acts of interns, residents or other hospital employees. Although most cases hold as noted in respect of the surgeon's liability, the latter, while in the operating room, would do well, because of his authority within such confines, to assume supervision and control of the nurse who is subject to his directives and duties during the operation.

The New York courts hold that an interne or resident is an employee of the hospital within the meaning of the Workmen's Compensation Act (Bernstein v. Beth Israel Hospital, 236 N. Y. 269); and the same ruling applies to staff nurse personnel who may become claimants as a result of injuries sustained during the course of their hospital employment. However, a special nurse is the agent of the patient and is not an employee of the hospital (Brown v. St. Vincent's Hospital, 226 N. Y. S. 317).

The New York courts have also held that although a hospital is legally liable for negligence in the performance of its administrative acts, e.g. to provide proper maintenance of its plant, hospital equipment and apparatus, to make general regulations for the conduct of the hospital, to employ competent nurses, etc., it is not responsible for the wrongful acts of physicians (which includes members of the interne and resident staff), or of staff nurses in the performance of their professional duties, unless there was negligence in their selection as members of the staff. This is on the theory that hospitals cannot be liable for professional negligence inasmuch as they do not treat patients, but engage only to procure nurses and medical personnel whose independent calling as far as the hospital is concerned, identifies them as independent contractors. (Matter of Renouf, 254 N. Y. 349; Necolayeff v. Genesee Hospital, 296 N. Y. 936.) It is the nature of the negligent act which determines the hospital's liability.

In this connection it is interesting to note that the New York State General Municipal Law as amended during the 1956 legislative session, now provides that malpractice indemnity coverage extends to protect from liability all physicians, interns and residents who do not receive any compensation from the patient. Receiving compensation from a municipally operated hospital by the personnel indicated, no longer will result in their loss of protection from liability when sued for malpractice. This protective enactment specifically applies to this class of personnel when serving in none other than municipally operated hospitals.

It is hazardous from the standpoint of being made civilly liable to lawsuit, for a physician to examine, treat, or operate upon a minor child without first obtaining consent of the parent or legal guardian, where the

consent could be waited for without danger to the patient. However, if consent is refused for surgical intervention where the presenting condition is threatening to the child's life, health or sanity, the Court may direct the surgery (in re Vasko, 263 N. Y. S. 552). A self-supporting infant, sufficiently mature to realize the dangers and benefits of a contemplated operation, who consents thereto after he has been fairly informed regarding his condition, cannot himself nor by his parents or legal guardian hold the doctor legally liable as far as the issue of consent is concerned. (Sullivan v. Montgomery, 279 N. Y. S. 575.)

Where an unknown and unconscious adult patient is brought to the hospital, the physician is not required to delay (for the purpose of securing consent) the rendering of the indicated life saving emergency surgery. Even though the patient be a minor, the presence of definite emergency implies constructive consent for the purpose of doing what is indicated to save life.

Where a defendant in a legal proceeding desires to have a plaintiff examined physically in order to verify plaintiff's alleged physical complaints, the physician employed by defendant to perform the physical examination will not be permitted to subject the plaintiff to such type procedures as cystoscopy, breathing tests, to eat a barium meal or to perform other hazardous or unpleasant procedures. (Carrig v. Oakes, 18 N. Y. S. 2nd 918.)

Any person who examines, diagnoses, treats and/or prescribes for human diseases, injuries and ailments engages in the "practice of medicine" within the New York State statute penalizing such practice without license, regardless of whether persons examined suffer from any ailments. (People v. Johnerson, 49 N. Y. S. 2nd 190.) Whoever is found guilty of such offense is adjudged as having committed an assault. X-ray treatment given by an x-ray technician at the hospital constitutes the "practice of medicine" and is in violation of Section 1250 (7) of the New York State Education Law (Leitch v. Mulcahy, 31 N. Y. S. 2nd 874).

The retention of possession by the physician of x-ray films taken of the patient, constitutes an important part of the latter's clinical record, and is as much a part of the patient's case history as slides made from the patient's tissue, patient's blood stains used in reading oxyhemoglobin scale for approximating hemoglobin contents of patient's blood, or any other preserved incidence which might justify the diagnosis of the condition for which the patient had originally received treatment. However, the question of ownership of x-ray films appears never to have been adjudicated by any high court except in one appellate tribunal in the State of Michigan. (McGarry v. Mercier, 262 N. W. 296.) Interesting enough, however, the New York courts have gone so far as to rule that ownership of ordinary photographic negatives, in the absence of an agreement otherwise, is vested in the operating photographer, though his use thereof may be restricted. (Lawrence v. Ylla, 55 N. Y. S. 2nd 343).

Hospitals as well as physicians have been subjected to lawsuits for civil damages by patients whose causes of action have spelled out an invasion of their right of privacy. Some examples of this intrusion are: examination, operation; any treatment carried out in the presence of others than the usual surgical or medical staff (Binns v. Vitagraph, 210 N. Y. 51); the taking of patient's photographs or x-rays without consent; or the subsequent use of such photographs; the televising of; or other showing or publication of any operative or other procedure performed on a patient; and last but not least, is the improper exposure of a patient's person which may cause him to suffer shame by having been exhibited to humiliation and embarrassment.

In an important decision (Griffin v. The New York State Medical Society, 11 N. Y. S. 2nd 109), the Court held that an article, even in a scientific publication, may be nothing more than some one's advertisement in disguise. Therefore, upon an allegation by the patient that photographs taken of him "before and after" and published without his consent as part of the printed article, the Court ruled that it was for the jury to decide whether the photographs were used solely for advertising or trade purposes—the latter in violation of the New York Civil Rights Law. It is well to bear in mind this

rule of law—(where physicians practising within New York State submit, in medical papers for publication, photographs of patients treated and described in their manuscripts)—that the unauthorized use or publication of photographs is one of the most common means of invasion of the right of privacy.

In the State of New York and in five other states, statutes have been enacted which require the physician or hospital attendant who treats or aids a person suffering from wounds inflicted by bullet, knife, or other weapon, or by some suspicious act of violence, to inform the police of the circumstances, name and description of the patient and other pertinent facts. (N. Y. Code of Criminal Procedure, Section 392, and Penal Law Section 1915.) In addition thereto, the States of Arizona and Iowa by statute, and the City of New York by ordinance in its Sanitary Code, Section 90, require similar reporting where the person treated or aided is suffering from injuries as a result of the commission of an abortion.

A physician cannot prescribe or administer to a woman whether pregnant or not, and cannot advise her to take any medicine or drug, nor can he use any instrument or device with intent to produce a miscarriage unless it is necessary to preserve life of the woman or the child. In order to escape being charged with the commission of the crime of abortion, unless and except to preserve life as noted, the burden of proof is upon the defendant physician so charged, to prove and establish the exception, which is that he terminated the pregnancy because of the necessity to save life. (People v. McGonegal, 17 N. Y. S. 147, and Section 80 New York Penal Law.) The New York courts have further held that the question whether the woman was pregnant is not the material element of the crime of abortion.

Where a physician may have been freed of the criminal charge of actual performance of an illgeal operation to produce an abortion, the New York courts will not interfere to set aside the determination of the Board of Regents which had previously suspended a physician's license to practice following his implication in an operation for abortion. (Genova v. Board of Regents, 74 N. Y. S. 2nd 729.)

If defendant physician, on being consulted as to a supposed pregnancy, furnished a drug with the statement that it would bring on menses, though actually the drug would not have caused an abortion, it is a violation of Section 1142 of the New York Penal Law, punishable as a misdemeanor. (People v. Hager, 168 N. Y. S. 183.)

Under New York Law, the courts hold that proof of breathing is not necessary, to consider the child as having been born alive. Although respiration could not be induced and the child was never heard to cry, nevertheless the fact that heart beats were perceptible is sufficient legal reason to state that the child was born alive. (Matter of Union Trust, 151 N. Y. S. 246.) The laws of the State of New York and of sixteen other states require that in stillbirths, the attending physician file both a birth and death certificate. In New York, and by coincidence in sixteen states, some of which are other than those whose laws promulgate the stillbirth regulations, it is unlawful and constitutes a crime, to falsify, to fail to file, or to refuse to give information for a certificate of birth or death.

New York, North and South Dakota and Oklahoma are the only four states which have enacted special statutes permitting a person the right to direct the manner in which any part of his own body which becomes separated during his lifetime, shall be disposed of. New York Penal Law, Section 2210, holds that it is an unauthorized mutilation of a human body to perform a post-mortem examination upon it without consent of the spouse or next of kin of the deceased. (Apostle v. Pappas, 277 N. Y. S. 400.) However, a direction contained in the last will of the deceased regarding the disposition of his remains is paramount authority therefor, and the deceased's permission for autopsy, cremation or burial of his remains is supreme, even though members of the family of the deceased interpose opposition. (In re Eichner's Estate, 18 N. Y. S. 2nd 573.) In New York and fourteen other states, statutes have been enacted which make it a criminal act to perform an un-

authorized autopsy. This is in addition to the civil liability for failure of consent therefor, as outlined in the preceding paragraph. New York, the District of Columbia, and four other states, have made it lawful by statute that an application for autopsy by an insurer (insurance company) provided it is made within a reasonable time after the death of the person, be granted for the purpose of determining the cause of death.

If a physician obtains a patient's consent for operation which was falsely represented to be minor, and then performs different serious surgery which results in the patient's death, the physician becomes guilty of an assault for the wrongful act, and recovery against him can be had under the Decedent Estate Law. (Griffin v. Bles, 195 N. Y. S. 654.)

Before statute laws were enacted in the various states and the courts functioned under common law as administered in England, communications between physician and patient were not privileged, and legal controversies were not modified or given exception on the basis of claim of ethical superiority. Statutory law, however, confers privilege and provides that confidential communications acquired by physicians and registered nurses in attendance upon a patient in a professional capacity, may not be divulged. Although the rule of privilege as enacted in seventeen states, has received criticism in some quarters particularly in respect of its alleged abuses in legal controversies involving insurance policies and in personal injury litigation, nevertheless the basic reasoning behind the protection of the privileged communication is that a physician is unable to give adequate treatment to his patient unless he is aware of all the facts surrounding the patient and any historical data referable thereto.

The protection of the privileged communication is of more than ordinary importance in psychiatric practice. Here the peculiar closeness of patient-physician relationship involves a confidence of the highest order. Were the patient to refrain from presenting full and frank discussion of his situation to the psychiatrist, out of fear of the possibility that the therapist may be forced to disclose what was made known to them in the privacy of the medical settings, such apprehensive withholdings would not only impede, but might destroy therapeutic efforts, and would engender in the patient the instinct of flight, rather than security from fear of exposure to physical or psychic dangers. The prohibition by law against such disclosure applies with equal force to patients as well as to their physicians, and extends as well to hospital records, electroencephalographic tracings and radiographs. No testimony from these sources may be introduced into evidence in any legal proceeding not involving a criminal act.

An intern or resident of a hospital staff, though not licensed to practice, is considered to be a "physician" within the meaning of the law (Black v. Ackerman, 63 N. Y. S. 2nd 445) and for this purpose is governed by the statute against revealing privileged communications. The physician cannot be asked to testify in a civil action as to the necessary information he had acquired during his attendance (whether in the hospital, doctor's office or patient's sick room), which knowledge enabled him to adequately treat the patient. But when the patient sues the physician for alleged malpractice, he waives the confidential physician-patient relationship in respect of information associated with the medical services regarding which the patient now complains, testifies about, or requests the physician to testify. (Ferrier v. Dare, 131 N. Y. S. 51); Albers v. Wilson, 195 N. Y. S. 145.)

(To be concluded in the next issue—January 1957.)

Neurotic Aversion to Kissing

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> Lord! I wonder what fool it was that first invented kissing.
>
> *Swift, "Polite Conversation"* (1738).

> The anatomical juxtaposition of two orbicular oris muscles in a state of contraction.
>
> *Henry Gibbons,* (1925).

There are neurotics who detest kissing and either follow Swift's lead, claming that the entire procedure is "distasteful," or line up behind Henry Gibbons to prove the "senselessness" of the act by reducing it to the status of an anatomical exercise. One such patient asked whether it would not be "more hygienic" to adopt the custom of rubbing noses to indicate affection. At one point or another, these neurotics have become blank to the emotional content of the kiss, or have transformed it negatively. The negative connotation is visible in some obsessional neurotics with infection fear; they allege that they avoid kissing because the contact transmits bacteria.

The hugs and kisses of adults, and especially of the mother, are distinguished as affectionate and non-inimical gestures by the child at an early age. "Non-inimical" is a more precise description than "loving," because the newborn baby—and the small child as well—misconstrues even harmless actions of the environment, believing them hostile. These misconceptions form the basis upon which the child builds "the septet of baby fears."* If a child persists in the fantasy that kissing means being "swallowed up" and "devoured," it can be expected that trouble will develop in this specific department in the future. At a later age, of course, these fears are rationalized.

Once kissing has been accepted as a positive emotional gesture, it also becomes a reassurance against fear. Children who kiss a great deal are not always merely affectionate; some of them are frightened. The paradox remains that kissing—originally misconstrued by the child as "being swallowed up"—*subsequently* becomes important as an assurance that he is loved, or at least accepted.

George Bernard Shaw, paraphrasing La Rochefoucauld, remarked in "Man and Superman," "There is always one who kisses and one who only allows the kiss." The difference is exactly that between being in love and allowing the other person to love.

It is also clear that kissing is psychologically more than "a juxtaposition of two orbicular oris muscles." It has some connection with friendly incorporation; the old unity of mother and child is re-established. This is visible in the moving pictures (which in this respect if in no other, assiduously copy life), where the standard kiss begins with the boy putting his arms around the girl, but becomes meaningful only when she in turn puts her arms around him. Here (from the boy's viewpoint) an active repetition of a passive infantile situation has taken place.

It is common knowledge that kissing may substitute for sex proper, as some kind of preparatory act, or may become a purpose in itself when used in perversions, as in fellatio and cunnilingus.** Perhaps this was the meaning of the famous phrase, "a paroxysmal kiss," which Henry Ward Beecher used in the Tilton-Beecher suit. It is also probable that Montaigne alluded to this substitution when he wrote in his "Essays," nearly four hundred years ago, of "the close-smacking kisses of youth, full of relish, greedy and sticky."

Since everything in this world can be misused, kisses can become tools of hypocrisy (Judas kisses): perfunctory kisses exchanged by relatives who hate each other; meaningless kisses exchanged as a conven-

*For elaboration, see the author's "The Basic Neurosis," Grune & Stratton, N. Y., 1949.

**For psychology of fellatio and cunnilingus, see the author's "Neurotic Counterfeit-sex," Grune & Stratton, N. Y., 1951.

tional greeting, etc. Kisses of a sexual tenderness constitute a special (non-hypocritical) subdivision. Nevertheless, the kiss remains what Robert Herrick declared it to be:

> What is a kisse? Why this, as some approve;
> the sure sweet—Sement, Glue and Lime of Love.

Kissing as a pleasure presupposes acceptance of the other person as "good"—meaning not dangerous—"food" of infancy. Many expressions in the language illustrate the link between love and edibility, a few familiar examples of which are "sweetheart," "honeybunch," "sugarpie."

In some neurotics, kissing is avoided because the infantile game is unconsciously perpetuated: "bad mother does not love child." In active repetition of an allegedly passively endured experience, these neurotics reverse the roles; remembering the mother's "rejection," they now do the rejecting themselves. But why do they express this rejection by refusing kisses, specifically? In a series of analyses I have encountered this peculiar reasoning: "My parents expected a good-night kiss from each child. These kisses were cold and perfunctory, and I hated the whole routine." By exaggerating the criticism, the kiss is rejected in toto. The child in the adult demonstrates, in an unconscious caricature, "Look how cold you were, Mother!"

Neurotic aversion to kissing plays a greater role than is generally known; many marital estrangements begin with distaste for kissing or refusal to kiss. Frequently the full neuroticism of the individual shows up in these situations, especially when preceded by a phase of sexual intercourse without tenderness.

Clinical Evaluation of Ritalin

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We are living in a difficult age where life is fast; war—and threats of war, high taxes, social and economical problems—there isn't anyone who at one time or another isn't under some terrific stress or strain. As a result, most of us although not physically or organically ill are on occasion depressed, fatigued, or otherwise feel low. It is in this largely neglected group of individuals where we could use some safe, psychomotor stimulant, one which would be effective yet not injurious to general health.

Then again there is a large group of incurable patients, the malignancy patients, the chronically ill, the aged, the convalescent, all of whom need some form of medication to give them a boost.

In the field of psychomotor stimulants, very few effective drugs have been developed since elixir iron quinine, strychnine and caffeine. About ten years ago Coramine and metrazol came into use but their benefit was limited. Then the amphetamine type of drug was developed. However, because of the depressing effects on appetite, the excessively high peaks of stimulation, the effect on blood pressure, and other side reactions, it also had a limited usefulness.

Now more than ever, because of the side effects of the newer types of drugs in modern medicine, there is a definite need for a more effective type of psychomotor stimulating drug, a safe drug to overcome mild depressions, fatigue, and the depressing, lethargic side effects of large amounts of barbiturates, chlorpromazine, antihistamines, anticonvulsants, and Rauwolfia.

Recently a new type of psychomotor stimulating drug, Ritalin® (methylphenidylacetate hydrochloride) was developed in Switzerland. After extensive animal and human experimentation and clinical trial,[1-22] it appeared to be the type of drug which would fit our present-day needs since it did not

have have the harsh, undesirable sympathomimetic action of the amphetamines or amphetamine-like agents.

Meier, et al.[1] found that Ritalin produced mental stimulation and a coordinated increase in motility in the rat, dog, mouse, and rabbit. According to these authors, the action of the drug lay somewhere between amphetamine and caffeine.

More than 3,000 psychiatric and nonpsychiatric patients have been treated clinically with Ritalin by over 200 investigators. Reports received from many of these clinicians indicate good to excellent response to the drug in overcoming chronic fatigue, sedation seen during Rauwolfia or chlorpromazine therapy, psychotic depressions, and depressions associated with menopause and nervous exhaustion.

Moreover, numerous studies have shown no adverse effect on peripheral blood, urine, liver, and kidney function. Except in rare instances, Ritalin does not depress appetite. Only one patient to date showed evidence of habituation without apparent harmful effect. This individual had a history of drug habitution.

The purpose of this study was to evaluate Ritalin in actual everyday general practice. It included 89 case studies on individuals whom I felt might benefit from the use of this drug, and who might be seen by any practicing physician.

In the study it was considered important to: 1) determine the effectiveness of the drug, 2) establish an average dosage, 3) look for side effects—immediate or delayed, 4) check toxicity and safety—with possible effects on blood pressure, the kidneys, the blood, blood sugar, or any other system in the body, 5) look for evidence of habituation, 6) establish indications for usage, 7) and suggest possible contraindications.

One can readily see that it is very difficult to evaluate the clinical results accurately in the use of such a type of drug, as there are no specific end results. So all one can do is evaluate the clinical response, subjectively, by determining whether or not these patients actually felt better after taking this drug. In private practice, my experience has been that patients readily will report any bad side effects, will discontinue a medication if they cannot tolerate it or if they feel they are not getting any results.

The patients used in this study fell into these groups: 1) the chronically ill and incurables—such as cancer patients, 2) the convalescing group, where the drug was used after illnesses or surgery to shorten their recovery period, and to give them a feeling of well-being, 3) tired, fatigued, depressed individuals who suffered from no organic disease but were worried and didn't feel well because of the strain or load of present-day living (this group made up by far the greatest number of patients in our series), 4) individuals who were on depressing drugs such as barbiturates, antihistamines, tranquilizing drugs, anticonvulsants, or Rauwolfia derivatives.

Each patient was given a thorough physical examination, fluoroscopy of the chest, complete blood count and urine analysis, blood pressure readings were taken, and any other procedures carried out when necessary to rule out any organic disease. Separate charts were kept on each individual patient, and included the following information: Diagnosis, age, daily dosage, duration of effects, duration on medication, pulse, blood pressure, blood count, weight, urinalysis, evidence of habituation, ability to overcome afternoon fatigue, side effects, tolerance to drugs, and personal comment.

At the beginning of the study there were about four patients who were unable to take the medication and refused to stay on the drug. One claimed she felt a peculiar sensation in the eyes. The other three were high strung, extremely nervous, or emotionally disturbed patients. Their complaints were gastric or rather bizarre symptoms, such as butterflies in the stomach. My experience with this and other stimulating drugs has been the same in this type of patient. They definitely require sedation and any drug which causes stimulation will cause them to feel worse. After eliminating this group of individuals it was found that almost all patients tolerated Ritalin well. A few patients taken off Ritalin because of nervousness were put back on the drug when the nervousness subsided and they had no further trouble with the drug.

The usual dosage was 10 to 20 mg. 2 or

3 times daily. In no case did I find it necessary to go above this dosage. The patient was instructed to start with 10 mg. twice or 3 times daily; and if there was no adequate response after a few days to increase the dosage to 20 mg. 2 or 3 times a day. In many older individuals I started with 5 mg. and increased it to 10 mg. 2 or 3 times a day.

About an hour after taking the drug most patients noted the stimulating effect of the drug and this stimulation carried them for 4 or 5 hours. It gave a plateau type of stimulation, no sudden peaks, or sudden letdown, but the effects gradually tapered off with no rebound. This stimulation manifests itself to the patient by a feeling of well-being and being alive, no euphoria, but fatigue disappears and he goes along all day without being tired. There isn't the jitteriness, headache, nervousness, or tense feeling which many patients feel when they take amphetamine drugs. It was noted in some that when bedtime came they felt alert and were unable to fall asleep. However, this was easily corrected by cutting the third dose in half, or if necessary eliminating the third dose and giving only two daily doses.

After eliminating the nervous individuals from the study, I did not find any cases in which the drug had an adverse effect on blood pressure, urine, blood count, pulse rate, or appetite. Many patients relaxed and felt so much better that their appetites increased and they put on weight.

In several of the carcinoma cases the results were excellent. W.P., a male patient with silicosis and a large inoperable carcinoma of the esophagus who was unable to swallow food was hospitalized. He had been ill for about 3 years, and had difficulty in breathing because of the silicosis. After he learned he had a tumor mass in the chest he became very depressed, especially after X-ray therapy was started. The patient cried and wouldn't even sit up in bed. After he was put on Ritalin he felt much more hopeful, quit crying, sat up in bed, shaved himself, and was in good spirits until the time of his death when he expired from a massive hemorrhage.

S.N., age 37, female, went into the hospital for a breast tumor. The lesion turned out to be a carcinoma and radical breast surgery had to be done. The day after she learned that she had a malignancy she became very morbid, cried, and was depressed and threatened to commit suicide. She was put on Ritalin and immediately her attitude changed. She felt better, stopped crying, and I experienced no difficulty with her after that. In fact she remained a month longer in the hospital to receive X-ray therapy but at no time did she manifest any symptoms of depression.

This winter our city had been polluted with a virus type of bronchitis. The untreated patients would go on for months with cough and other symptoms. Many of them ended up with pneumonia. With such a lingering illness or infection they became rundown, anemic, and didn't seem to get back their normal feeling of well-being. Those whose blood counts were low were given medications to correct their anemias. Simultaneously Ritalin was given and they felt much better and could carry on their work even during the time their counts were approaching normal. The others also recovered more rapidly and felt better when they were put on Ritalin.

The length of time patients were kept on the drug varied, depending on each patient's needs; some were on it only a few weeks while others took the drug for a year. The bulk of the patients who were on the drug because of depression, worry, or pressures of today's living, merely took it until they felt better. If they had a recurrence of a "low-down" feeling, they took the drug until they felt better. Some older individuals who had physical impairment because or degenerative changes of old age were kept on the drug for long periods of time.

In no instance in this series did any one develop a tolerance to the drug or show any evidence of habituation when the drug was discontinued. Although allergy to any drug shows up sooner or later, there was no evidence of any allergic manifestations in any of the patients in this study.

My experience uncovered no contraindications for the drug when it was used in the established dosages, except in the mental, extremely nervous, and emotionally disturbed individuals where the drug is not indicated.

Summary

Ritalin, a new type of psychomotor stimulant, was clinically evaluated in a series of 89 cases in actual private practice. These patients represented the types of cases which might come into any doctor's office for treatment.

The patients selected fell into four groups: the chronically ill and incurables, the convalescing group, the "low" patients, depressed because of pressure of present-day living, and the group who were on medications which caused depressed states.

The average established dosage of the drug was found to be 10 to 20 mg. 2 or 3 times daily. In older patients, many required only 5 to 10 mg. 2 or 3 times daily.

The drug gave a plateau type of stimulation, smooth onset, with no euphoria, no tachycardia, no headaches, no gastric symptoms, no high feeling, no tenseness or nervousness, such as is produced by the amphetamine type of drugs. The effect lasted about four hours, gave the patient a feeling of well-being and that life was worth living. Their worries seemed to disappear; they were alert, fatigue disappeared, and they could go all day without tiring. The effects gradually disappeared with no extreme let-down or rebound effect.

This was important in the largest common group—the neglected group of patients who didn't feel well, who had nothing organically wrong but were under every day pressures. They have been neglected because up until now there was no type of drug one could safely use on these people. They don't seem to do well on sedative type of drugs.

The side effects of Ritalin are minimal. There were some individuals who were alert and wide awake at bedtime after taking the drug late in the day. This is not serious and

SUMMARY OF RITALIN STUDY

Group	Diagnosis	No. of Patients	Daily Dose (mg.)	Duration of Rx (weeks)	Patient's Response: poor	Patient's Response: good	Patient's Response: excellent	Side Effects	Evidence of Habituation
I	Chronically ill, including aged and incurables	26	5-20 b.i.d. or t.i.d.	8- 4 wks 7- 8 wks 7-12 wks 3-26 wks 1-52 wks	1	3	22	2—too alert at bedtime	None
II	Convalescing group (post surgical and medical)	18	10-20 b.i.d. or t.i.d.	7- 4 wks 6- 8 wks 5-12 wks			18	4—too alert at bedtime	None
III	Counteract oversedation to barbiturates, antihistamines, anticonvulsants, or Rauwolfia preparations	6	10-20 b.i.d.	2- 4 wks 3-12 wks 1-26 wks	3	1	2	None	None
IV	Fatigued, tired, depressed due to pressures of modern day living	39	Children 5-10 b.i.d. or t.i.d. Adults 10-20 b.i.d.	4- 1 wk 15- 4 wks 11- 8 wks 7-12 wks 2-26 wks	5	7	27	6—overstimulation or jitteriness 4—too alert at bedtime 1—bizarre eye symptoms	None None
	TOTAL	89			9	11	69		

can be corrected easily by cutting the third dose in half, or eliminating it entirely.

The failures in this series of cases (where patients had to be taken off the drug because they felt they could not tolerate it) made up only a very small percentage of the total. They complained of bizarre symptoms, such as butterflies in the stomach, pressure on eyes, and hyperstimulation. These cases appeared early in the investigation.

However, after it was noted that these individuals were high-strung, extremely nervous, emotionally disturbed, borderline psychotic and psychoneurotic, I realized that any form of stimulation will make them worse, and more nervous. Many of these patients when they calmed down with sedation or tranquilizing drugs were put on Ritalin again and they tolerated the drug well. When this group was eliminated in the later studies no instance was found where it was necessary to take any patient off the drug.

The work showed that the drug had no effect on blood pressure, the blood count, urine or blood sugar, did not depress the appetite, and produced no tachycardia. There was no evidence of any allergic manifestations in any of the cases.

Patients were kept on this drug as long as their conditions warranted usage of the drug. These periods varied from a week to a year; however, in no instance did any patient develop tolerance to the drug, or show any evidence of habituation when they were taken off the drug.

Other than emotionally disturbed, or extremely nervous patients, no other conditions were found in which the drug was contraindicated.

Author's Note: The Ritalin® used in this study was kindly supplied by Ciba Pharmaceutical Products, Inc., Summit, N. J.

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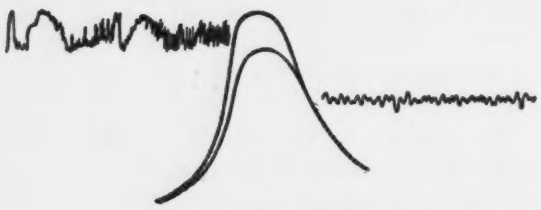

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1. Doyle, P. J., and Livingston, S.: J. Pediat. *43*:413 (Oct.) 1953.
2. Livingston, S., and Petersen, D.: To be published.
3. Pence, L. M.: Texas State J. Med. *50*:290 (May) 1954.

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